23andme – Genetic Portrait of an HSP
A Long-Lived "Highly Sensitive Person."

Personal genotyping report by **Stephen Arthur**, Vancouver Canada, 2013

(SNP = single nucleotide polymorphism)

In addition to finding that I have generally a low risk for the diseases of aging, I discovered that I also have specific longevity from a rare SNP in FOXO3A(CC). This SNP reduces the insulin-related mitochondrial pathway that "triggers" the diseases of aging, as described brilliantly by biochemist Nick Lane in his book "Life Ascending." According to Lane's hypothesis, the slightly higher risk that I have for a few cancer types will likely be erased by my F0X03A longevity polymorphism.

The twist to this gift of longevity comes from a combination of my SNPs that produce deficits in several of my neurotransmitter and neurohormone pathways. In short, I'll probably live a long but miserable life. Six of my SNPs that affect the central nervous system (CNS) explain two things about me. They reveal the mechanisms of what makes me a "Highly Sensitive Person" (HSP), as described in Elaine Aaron's classic book of that name. And they reveal why I have to consciously control my alcohol consumption. It turns out that I have 3 different SNPs that increase my risk for alcoholism by a factor of about 14 times. These include a polymorphism in the dopamine D2 receptor, in the opioid Mu receptor, and in a dopamine metabolizing gene. This tells me that I was born an alcoholic and should take that seriously.

As to being HSP, a polymorphism in an enzyme (COMPT) for inactivating both hormonal adrenaline in the CNS and the neurotransmitter noradrenaline leads to increased sensory and emotional pain. The same enzyme polymorphism has a secondary effect, which is to decrease responses to hormonal endorphins, further increasing pain sensitivity. This is further compounded by the previously mentioned variant in the Mu-opiod receptor. Finally, reduced serotonin in my brain, due to another slowed-down enzyme (tryptophan hydroxylase), promotes an anxious personality. All these traits add up to explain what I already suspected about myself. And they show that I was largely born this way, rather than learned to be this way from the serious grief and trauma of my youth.

Fortunately, one more neurohormone SNP provides a saving grace or antidote to the sort of "shadow syndrome" described above. I have high oxytocin, the "love hormone", which can reduce anxiety and cortisol, and can make me prosocial, responsive, and empathetic. This is the empathy side of the classical HSP.

When this portrait of traits includes my second, different SNP in the enzyme affecting dopamine levels in the prefrontal cortex of my brain, we seem to have a paragon of the classically described HSP. This SNP gifts me with greater attentiveness and curiosity, and makes me a "worrier not a warrior."

Much of this information I found by uploading my raw data file to SNPedia. This way I was also able to discern that my blood pressure is affected by sodium, so it is worth continuing to avoid salt. And I was able to clear up a mystery about being a fast metabolizer of caffeine even though it affects me strongly and lasts a very long time. It turns out that I have two other SNPs that undercut the fast metabolizing SNP. One SNP causes slow metabolism, and the other SNP makes it slower still, reducing the maximum rate of metabolism down as low as 5 percent of normal (Vmax 5%).

Mail your saliva and $100 to 23andme.com and find out about YOURSELF from genome chips, a service that would have probably cost you $100,000 only a few years ago.

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